NASA CONTRACTOR REPORT

NASA CR-176

THE EFFECT OF BEDREST ON VARIOUS PARAMETERS OF PHYSIOLOGICAL FUNCTION

PART VI. THE EFFECT OF THE PERFORMANCE OF PERIODIC FLACK MANEUVERS ON PREVENTING CARDIOVASCULAR DECONDITIONING OF BEDREST

by Fred B. Vogt, David Cardus, Carlos Vallbona, and William A. Spencer

Prepared under Contract No. NAS 9-1461 *by*
TEXAS INSTITUTE FOR REHABILITATION AND RESEARCH
Houston, Texas
for

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By F.B. Vogt, M.D., D.Cardus, M.D., C. Vallbona, M.D., and W.A. Spencer, M.D.

ABSTRACT

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Six subjects participated in two 3-day bedrest periods: the first period consisted of bedrest alone, the second period consisted of bedrest plus periodic Flack maneuvers, and the periods were separated by 4 days to allow for recovery. At the end of the first period, 3 of 6 subjects experienced convulsive syncope while performing the Flack maneuver in the 70 degree tilt position. The subjects had not recovered at the start of the second period of bedrest. Repeated Flack maneuvers during bedrest did not prevent manifestation of orthostatism found with bedrest.

FOREWORD

This study is a part of a NASA investigation of the effect of bedrest on various parameters of physiological function. It was sponsored by NASA Manned Spacecraft Center under Contract NAS-9-1461, with Dr. Lawrence F. Dietlein, Chief, Space Medicine Branch serving as Technical Monitor.

This study was conducted in the Immobilization Study Unit of the Texas Institute for Rehabilitation and Research, the Texas Medical Center. The authors are affiliated with Baylor University College of Medicine as follows: Dr. Vogt, Department of Rehabilitation; Dr. Cardus, Departments of Rehabilitation and Physiology; Dr. Vallbona, Departments of Rehabilitation Physiology, and Pediatrics; and Dr. Spencer, Department of Rehabilitation.

The authors wish to acknowledge the assistance of Mrs. Patsy Dominy and Mrs. Carolyn Caldwell in the typing of this manuscript; Mrs. Lavaun Shropshire, Mr. Troy Allen, and Mr. Tom Townsend in the preparation of the data for this report; and the Bioinstrumentation Section of the Space Medicine Branch, Manned Spacecraft Center for the instrumentation used in this study.

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SUMMARY

An experimental study was performed at the Texas Institute for Rehabilitation and Research in March of 1963 to evaluate the safety and feasibility of the use of repeated Flack maneuvers in preventing the cardiovascular deconditioning resulting from the hypodynamic experimental situation of bedrest. Six young adult male subjects participated in two 3-day periods of bedrest; the first period was bedrest only, while the second period had added the performance of Flack maneuvers every 30 minutes for 30 seconds from 6 a.m. to 12 midnight daily. Bedrest periods were separated by a 4-day recovery period.

At the end of the first bedrest period, 3 of 6 subjects experienced convulsive syncope while performing the Flack maneuvers in a 70 degree tilt position. One of the 3 subjects experienced cardiac asystole for a period of 8 seconds, but recovered spontaneously upon being tilted down. Heart rate changes with tilt indicated that the subjects had not recovered at the start of the second bedrest period. Cardiovascular deconditioning was observed after the second period of bedrest to which repeated Flack maneuvers were added.

INTRODUCTION

Orthostatic changes in heart rate and blood pressure have been noted to occur in association with prolonged bedrest[1,2,3,4] and water immersion tests.[5,6,7,8,9,10] A marked increase in heart rate, decrease in blood pressure, and in some cases syncope are manifestations of the cardiovascular deconditioning that occurs in association with tilting a person to the upright position after prolonged bedrest or water immersion. Since the conditions of bedrest and water immersion

simulate some of the conditions associated with space flight, it has been predicted that cardiovascular deconditioning could occur in association with space flights. The observation of postural changes in heart rate and blood pressure on Astronaut Schirra[11] after his relatively short orbital flight, and the failure to register a blood pressure measurement on Astronaut Cooper[12] while he stood still on the deck of the recovery carrier have added strength to this prediction.

A mechanism contributing to the development of cardiovascular deconditioning during recumbency or weightlessness is described simply as follows. During physical activity on earth, changes in vascular pressure resulting from the interaction of gravitational and muscular forces in the dependent parts of the body may be partially or totally responsible for the normal maintenance of compensatory reflexes that regulate the cardiovascular system and the volume and distribution of fluids in the different compartments of the body. The absence of gravitational force vectors in recumbency result in a redistribution of the hydrostatic pressure along the cardiovascular axis. This may be the initial cause of the chain of events that leads to the orthostatic changes in blood pressure and heart rate that can be observed in these two circumstances.

Since the Flack maneuver is known to produce marked pressure changes in both the venous and arterial systems, its use was proposed as a possible means to prevent the development of cardiovascular deconditioning. Such a procedure could be included in a space flight only after its beneficial effect and safety had been proven. It was the purpose of this study, performed in March, 1963, at the Texas Institute for Rehabilitation and Research, to evaluate the safety and feasibility of the use of the Flack maneuver in preventing the cardiovascular deconditioning resulting from the hypodynamic experimental situation of bedrest.

METHOD

Six male subjects, ranging from age 21 to 30 years, were selected after initial medical histories and physical examinations were performed to rule out any contraindications to their participation in the study. Two of the subjects were temporarily out of college; one had just completed college; one had been employed part-time in a newspaper office; one was a NASA engineering employee; and one was a cooperative student temporarily located at NASA. Five of the subjects participated in both the bedrest (with and without periodic Flack maneuvers) periods; but one subject, O.G., participated in only the bedrest period of study.

The experimental design provided for two bedrest immobilization periods using the same group of six subjects. The purpose of the first period was to collect control data on each subject to define the deconditioning that resulted from bedrest immobilization. The second bedrest immobilization study was then performed in an identical manner to the first, except for the addition of periodic Flack maneuvers. During this second immobilization period, the subjects were required to perform a Flack maneuver for a duration of 30 seconds, every 30 minutes, during the 6 a.m.

to 12 midnight period. Comparison of the data obtained during bedrest with those obtained during bedrest plus the Flack maneuver would thus provide information on the effect of adding the Flack maneuver. Figure 1 shows the time schedule of the studies performed.

The subjects were kept at strict bedrest during the 72-hour bedrest periods. They were allowed to turn over in bed, use one pillow under their heads, and feed themselves. They were not allowed to sit in bed, raise their arms and legs above head level, or get up for bathroom privileges. They were fed 2200-2500 calorie low residue diets to control the frequency of defecation. During the study they were allowed to drink fluid ad libitum.

A recording of the electrocardiogram, impedance pneumogram, and cuff-microphone blood pressure was made for a duration of 3 to 5 minutes, every 2 hours, on each subject during the periods of recumbency.

Prior to, and immediately after the 72-hour immobilization periods, tilt table studies were done to measure the orthostatic changes that occur with tilting of the subjects. Figure 2 shows the tilt procedure carried out in the pre-immobilization and post-immobilization studies. A motorized tilt table, which tilted the subjects from 0 to 70 degrees in approximately 35 seconds, was used. The subjects were supported by a pelvic harness system. The electrocardiogram, impedance pneumogram, and cuff-microphone blood pressure (cycled every 30 seconds) on each subject during tilting were recorded with a Physiograph.

During the second period of recumbency, the Flack maneuver was used as a conditioning procedure. Because of marked cardiovascular responses that occur with its performance, it was also used during the tilt evaluation as a stress test or provocative test to detect latent cardiovascular deconditioning. The Flack maneuver was performed with a Flack tester designed by the personnel of NASA's Bioinstrumentation Section. The device consisted of a cylindrical whistle-type apparatus with a spring-loaded plunger that provides 40 mm of mercury load to expiration. A small air leak was provided in the apparatus to assure that the pressure was transmitted from the pulmonary system rather than simply from cheek muscle action. A pressure recording device was attached to the Flack tester to register the actual pressure exerted during the maneuver. The subject performed the Flack maneuver by first taking a deep breath, and then blowing into the device to maintain the intra-oral pressure constant at 40 mm Hg for a duration which averaged 30 seconds. The duration was shorter if the procedure was interrupted because of impending syncope, and longer if tolerated by the subject.

In the tilt studies of the first bedrest period, the subjects were tilted down from the 70° position to the 0° position if they experienced syncope or convulsions. The subjects were observed very carefully during the tilt studies of the second period and were tilted down immediately if there were signs or complaints of impending syncope.

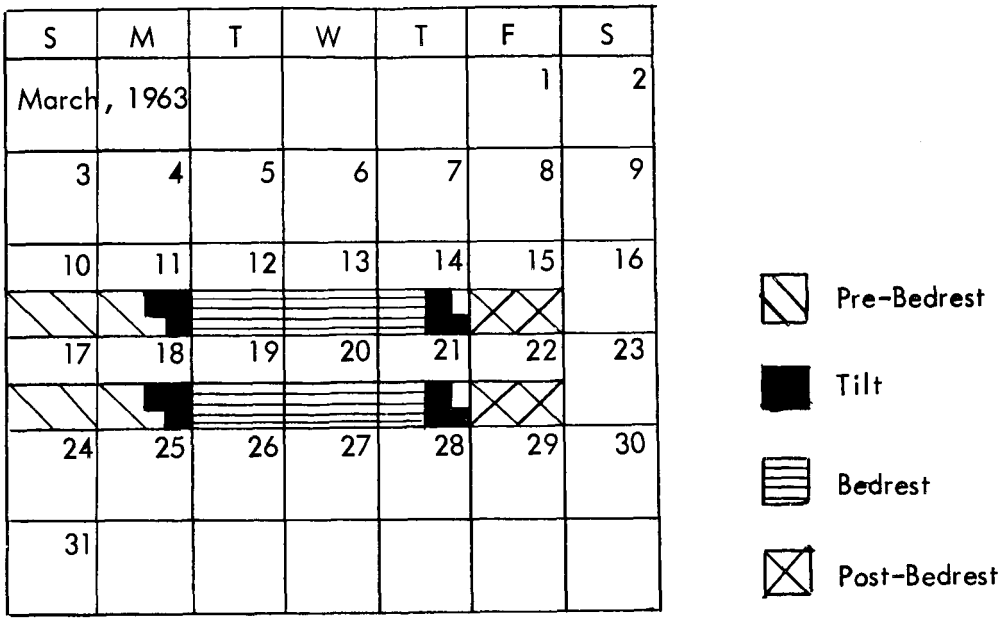
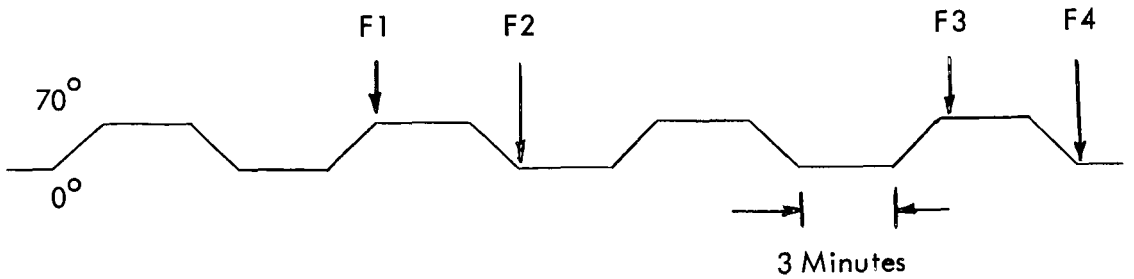


Figure 1. Periods of experiment.



F1, F2, F3, F4 = Time of performance of Flack test

Figure 2. Time schedule of tilting and Flack testing.

Bioinstrumentation equipment and techniques already available at the Texas Institute for Rehabilitation and Research were used in the experimental study. The electrocardiogram was obtained using NASA electrodes[13] attached at the M-X[14] position. Blood pressure was obtained by a cuff-microphone technique[15] from the left arm of the subjects. Respiration was measured by an AC-coupled impedance pneumograph[16] with NASA electrodes attached transthoracically at the sixth intercostal space in the mid-axillary line. Recordings were made on a Physiograph[17] at the time of monitoring, and the records were analyzed later. Figure 3 shows a sample record of the data obtained during the study. The electrodes and blood pressure cuff were attached to the subjects continuously throughout the immobilization period.

RESULTS

Figures 4 through 14 show the plots of the blood pressure, pulse pressure (systolic minus diastolic pressure), respiration rate, and heart rate obtained during the immobilization periods. The heart rate was determined by counting QRS complexes in three separate 30-second intervals, averaging these values, and then converting to beats per minute. The respiration rate was determined by counting respiratory excursions for a 1-minute period in the mid-portion of the record. All blood pressure measurements (six to eight) obtained during the recording interval were used to determine the mean value which is reported. A separate graphic presentation of the variables recorded in this experiment is presented in these figures for the individual subjects for each period of recumbency.

A very definite circadian variation in heart rate and respiratory rate is seen in subject R.S. Subject T.G. shows a circadian variation in his heart rate but the pattern of change of his respiratory rate is not as distinct. He also demonstrates an upward trend in heart rate in the second study period. He further shows a lower pulse pressure in the second period compared to the first. There were no apparent patterns of change in the vital signs of subject B.S. Subject J.P. did not show as distinct a circadian variation in his heart rate, but his differential cuff pressure (pulse pressure) showed a similar pattern of change in the two periods, with high and low values occurring at corresponding times. There was a suggestive upward trend of the blood pressure in subject K.W. in the first period of study. Subject O.G. showed a circadian variation in heart rate and respiratory rate that paralleled each other.

The variation in measurements taken at different times on the group showed considerable range of values both for the individuals and for the group as a whole. Values for respiratory rate ranged from 6 to 22 breaths per minute, for heart rate from 44 to 92 beats per minute, systolic blood pressure 86 to 144 mm Hg, and diastolic blood pressure from 44 to 94 mm Hg.

The tilt table procedures performed prior to the first immobilization period showed minimal changes of blood pressure and heart rate when the upright position

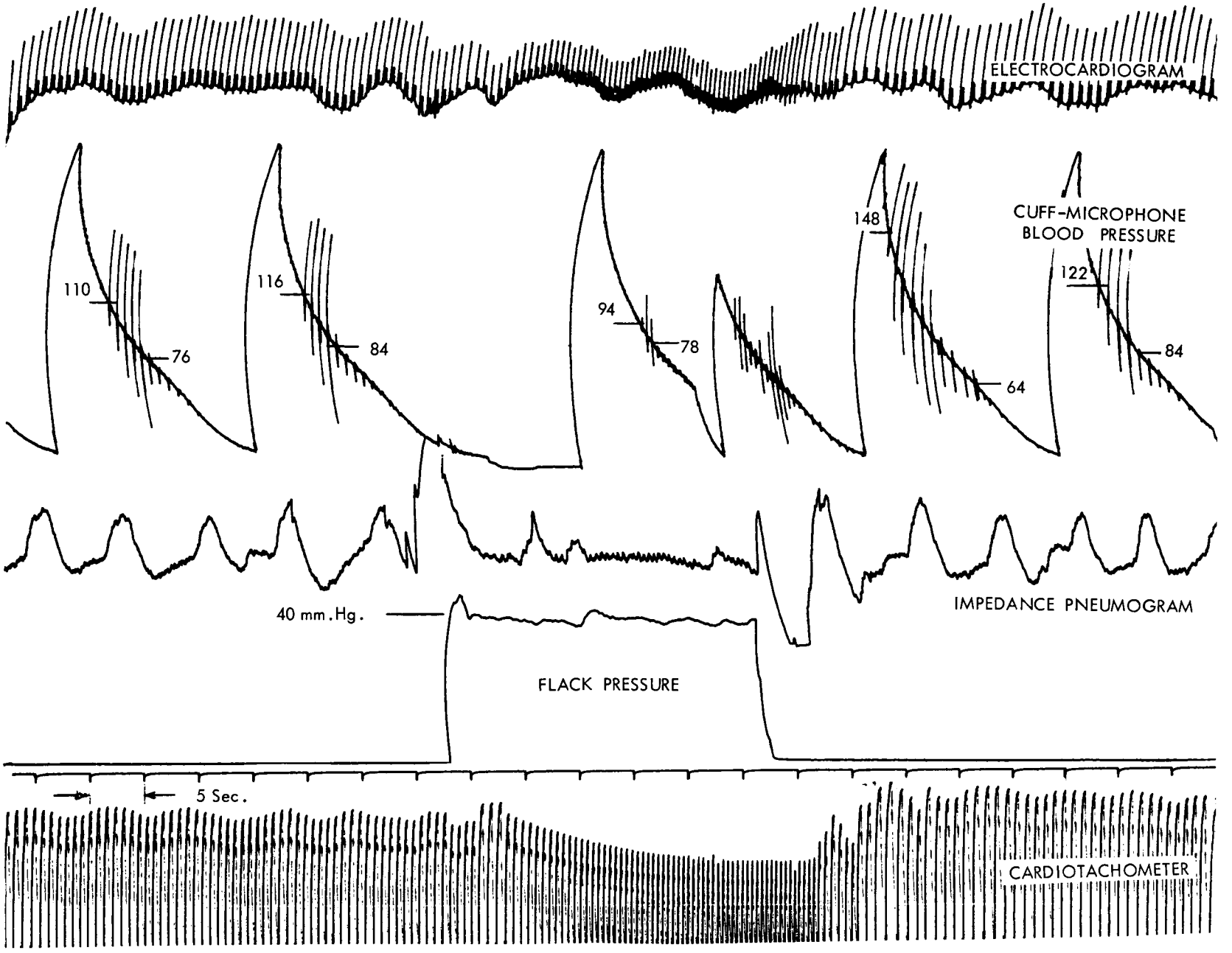
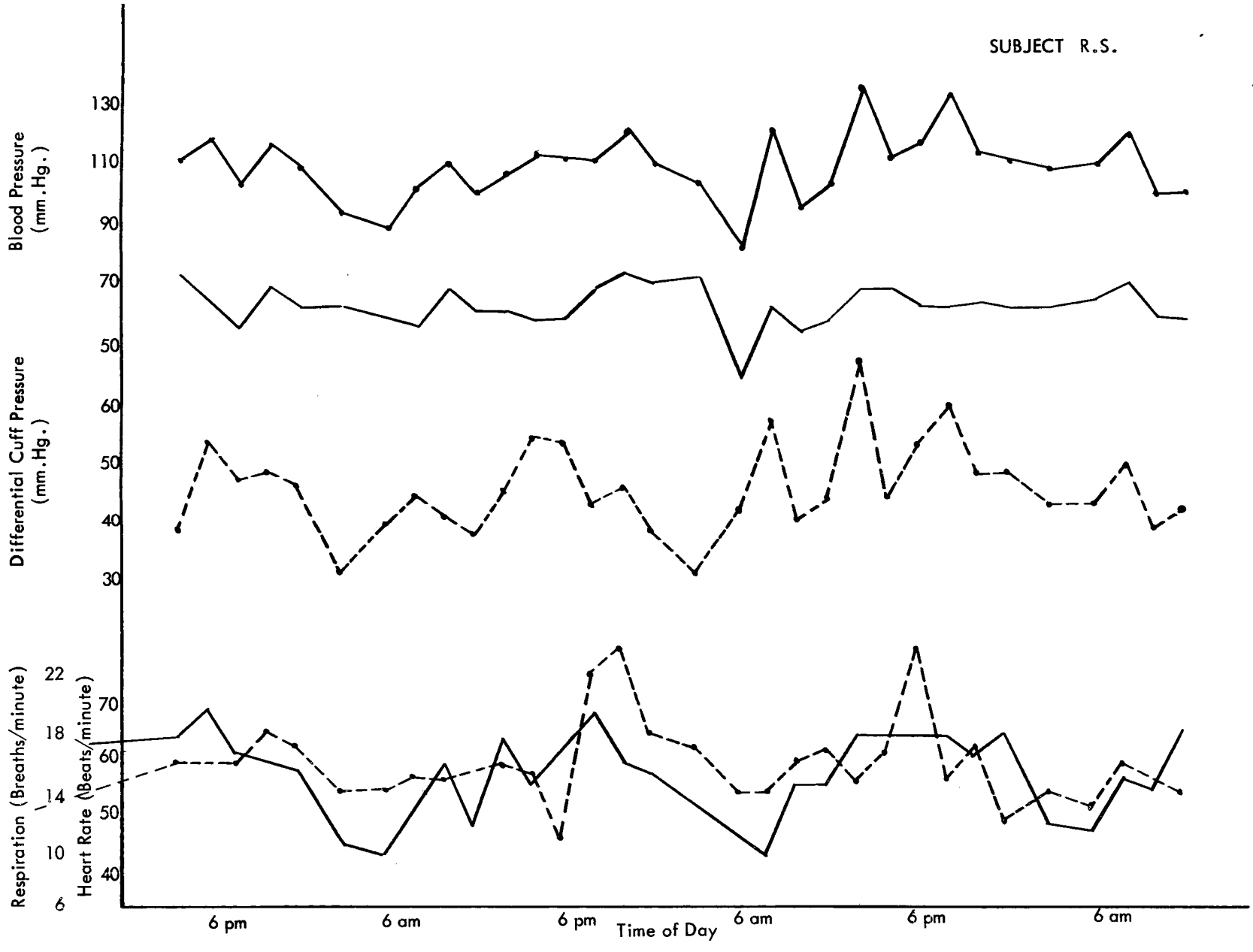
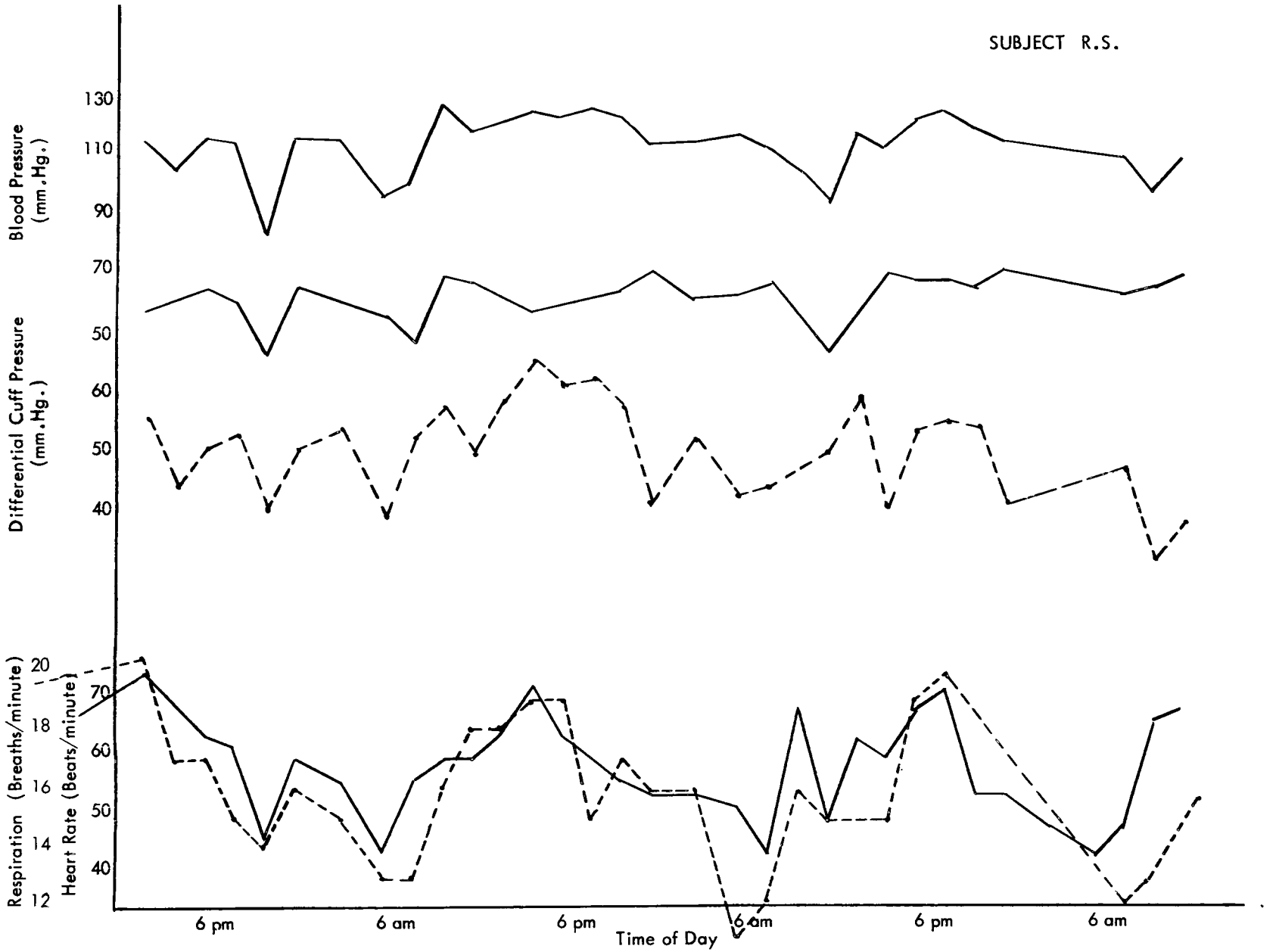


Figure 3. Sample Physiograph record.

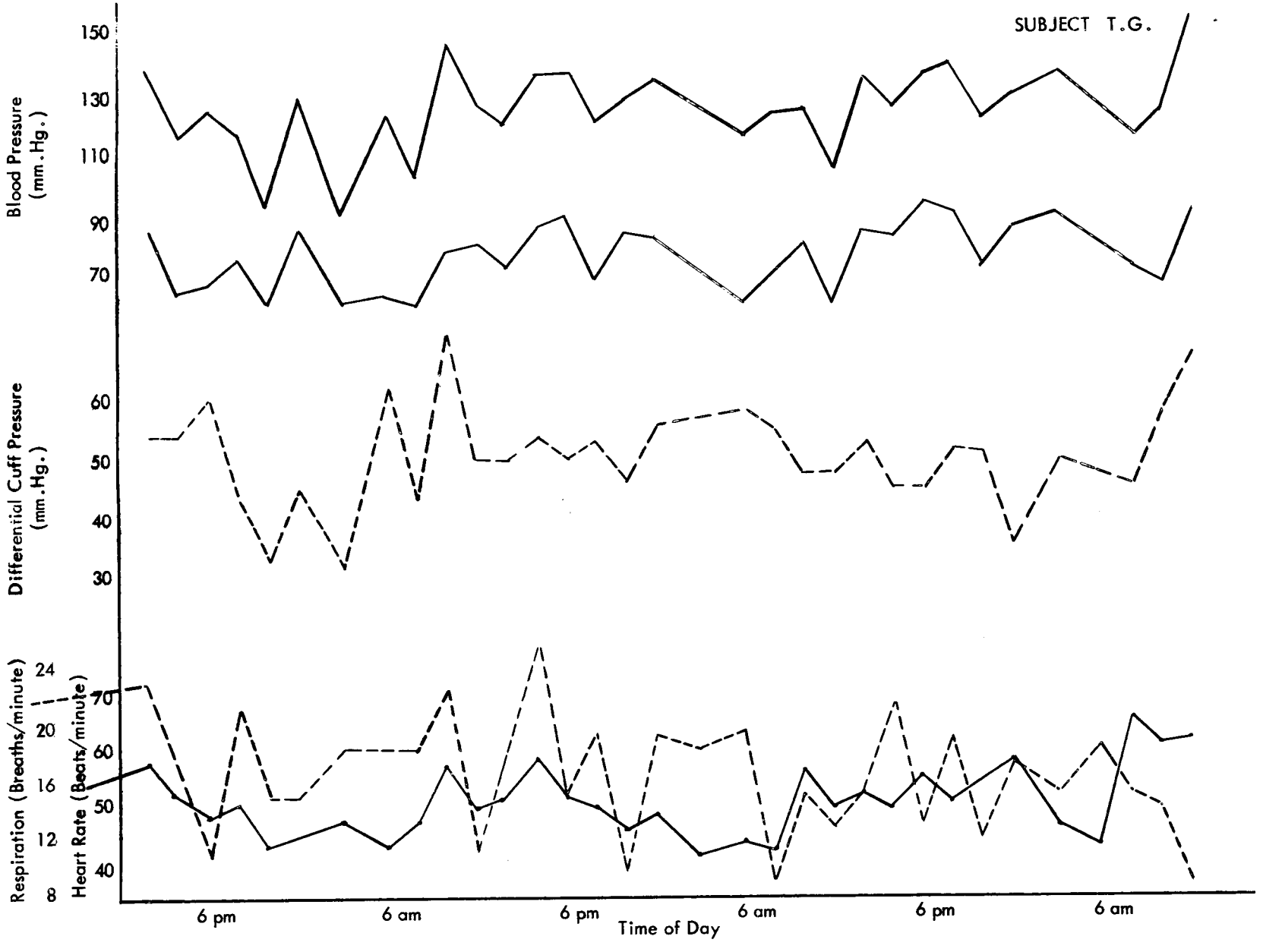


Subject R.S.

Figure 4. Vital signs -- 72-hour bedrest study.

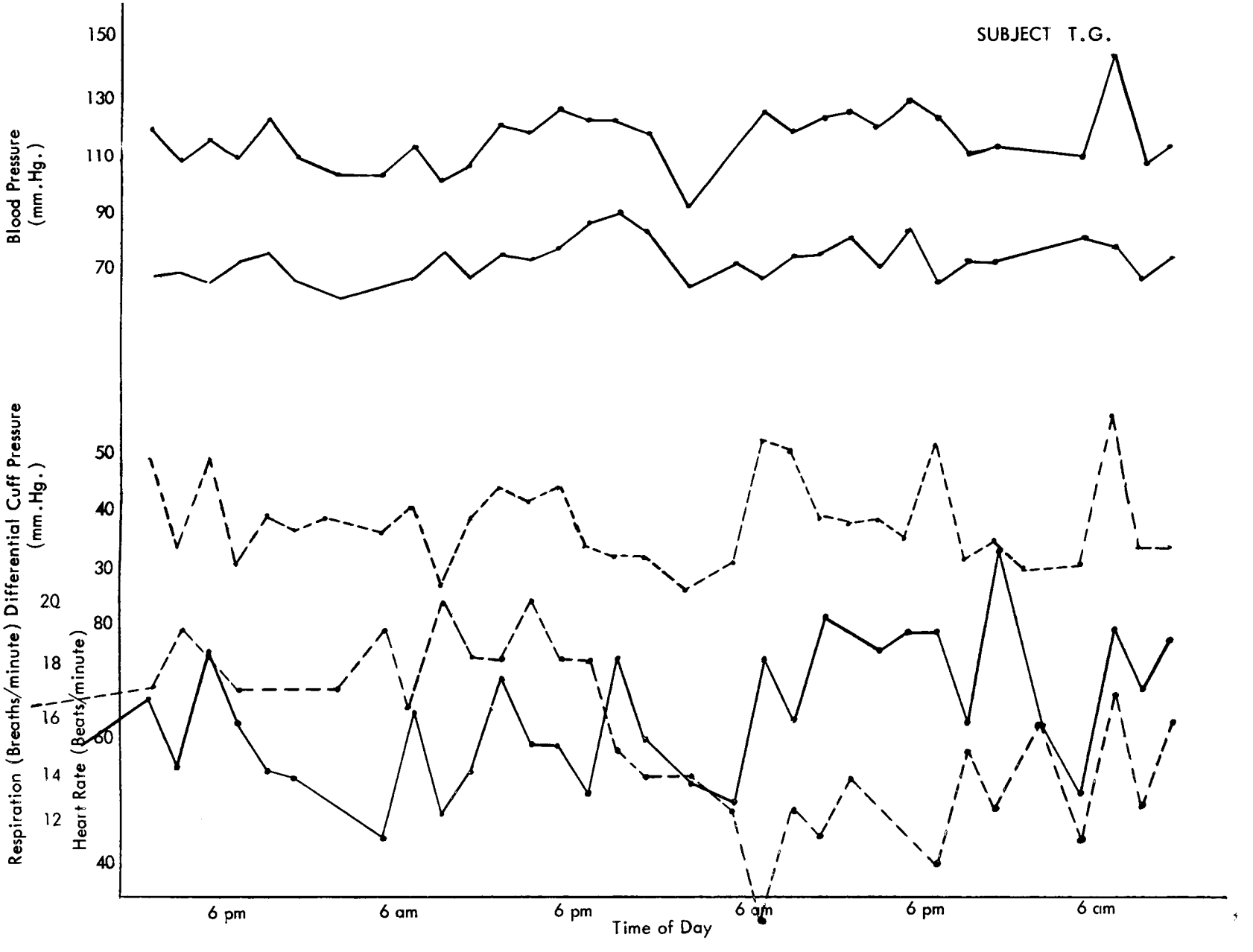


Subject R.S. Figure 5. Vital signs -- 72-hour bedrest study with the Flack Maneuver.

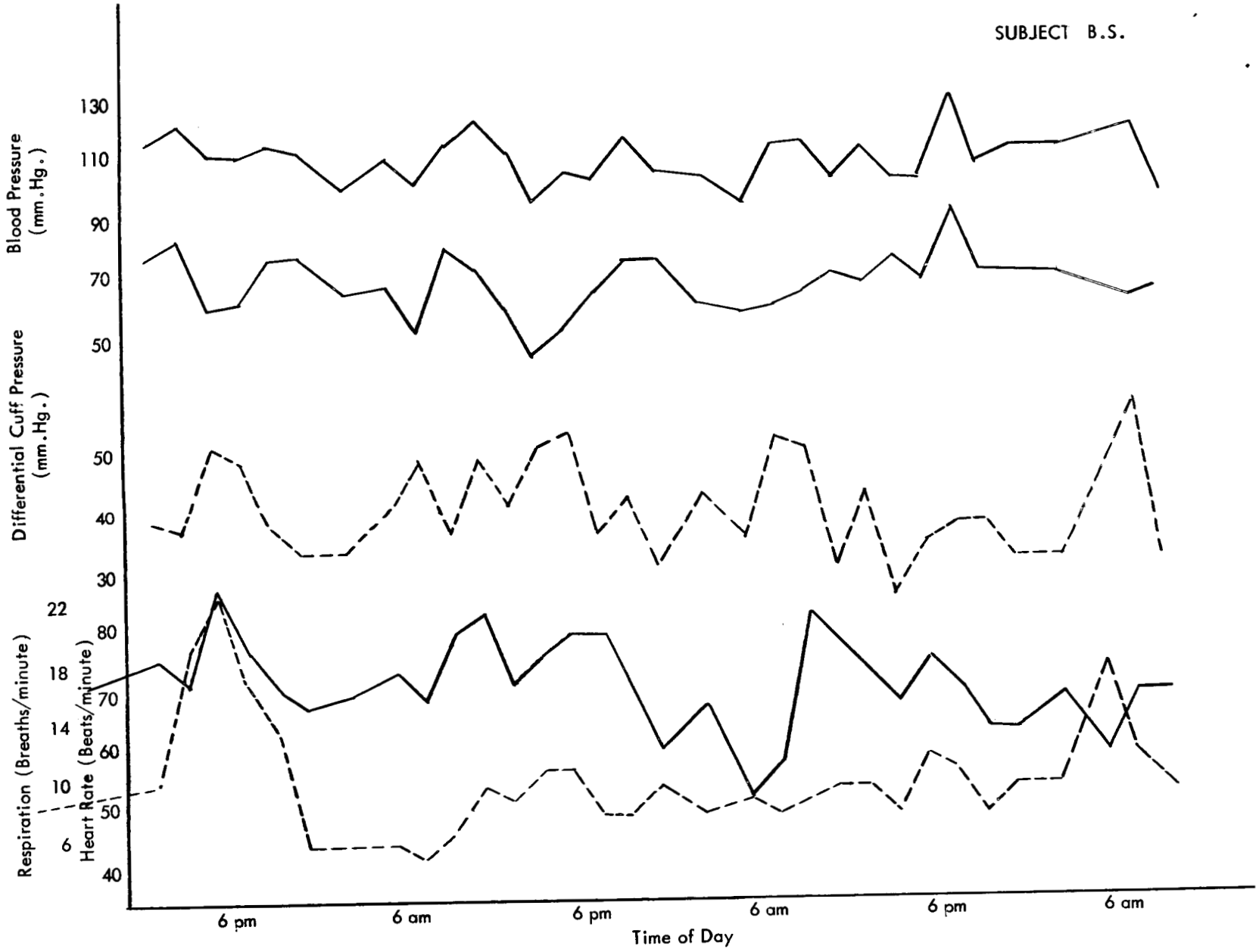


Subject T.G.

Figure 6. Vital signs -- 72-hour bedrest study.

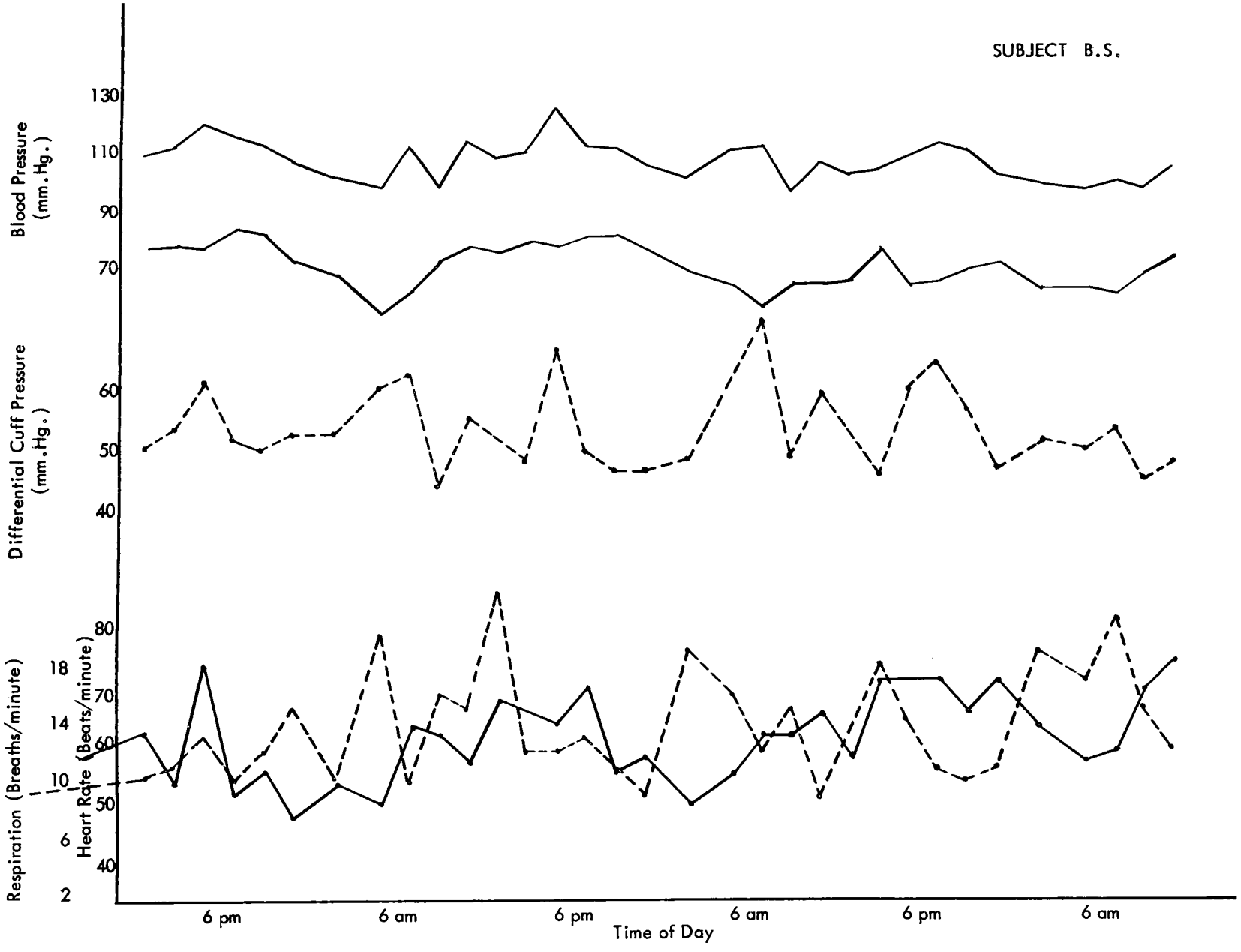


Subject T.G. Figure 7. Vital signs -- 72-hour bedrest study with the Flack Maneuver.

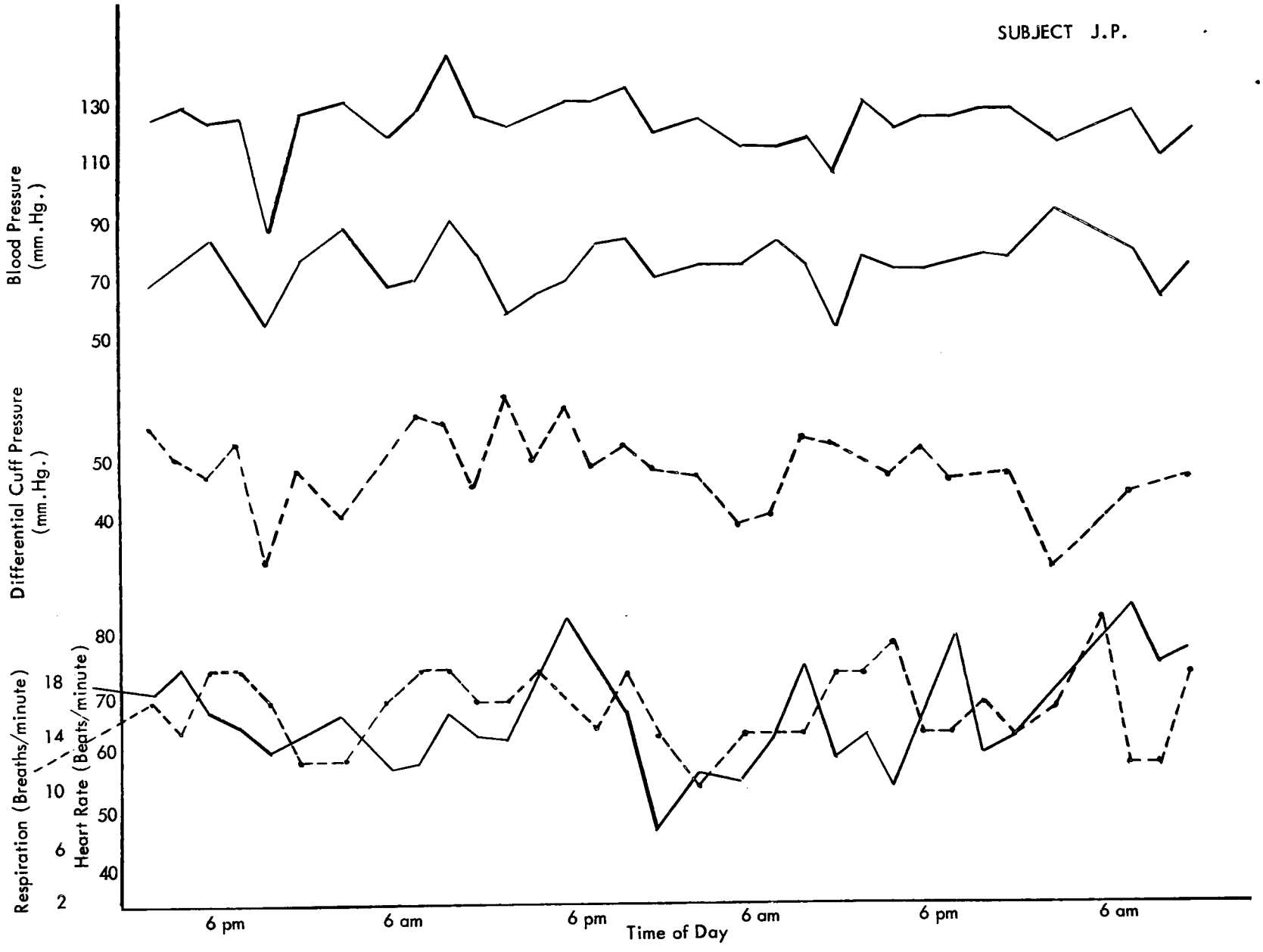


Subject B.S.

Figure 8. Vital signs -- 72-hour bedrest study.

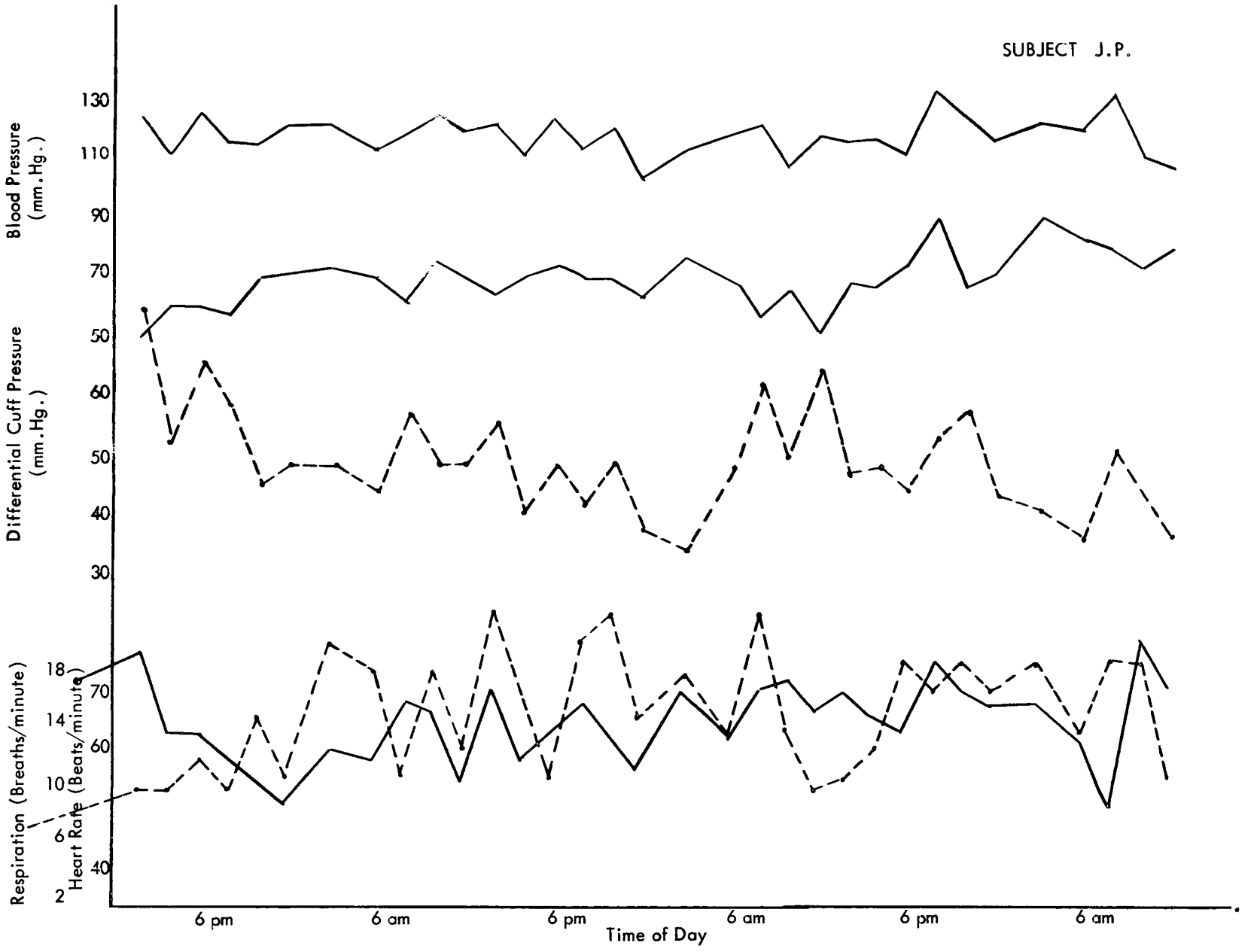


Subject B.S. Figure 9. Vital signs -- 72-hour bedrest study with the Flack Maneuver.

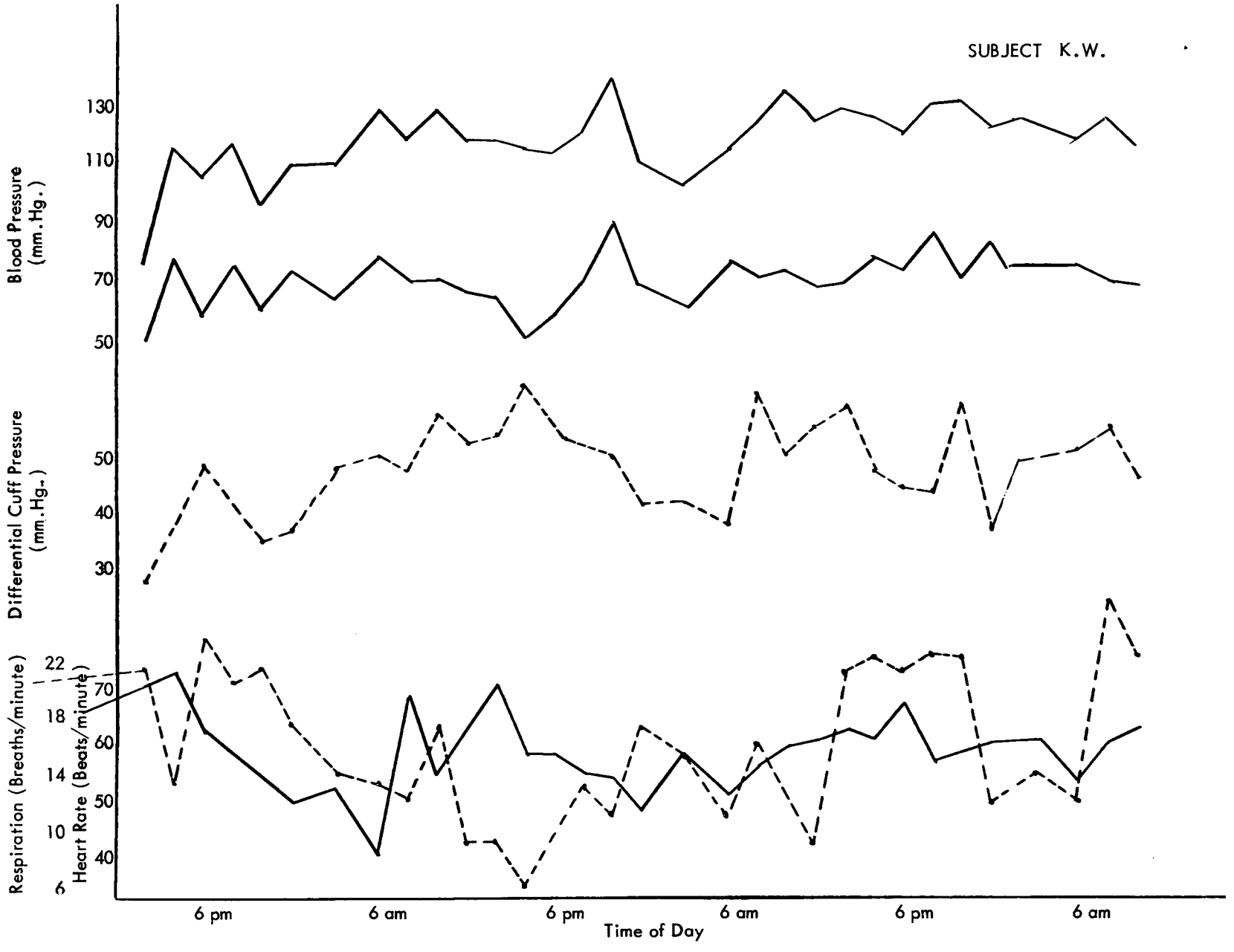


Subject J.P.

Figure 10. Vital signs -- 72-hour bedrest study.

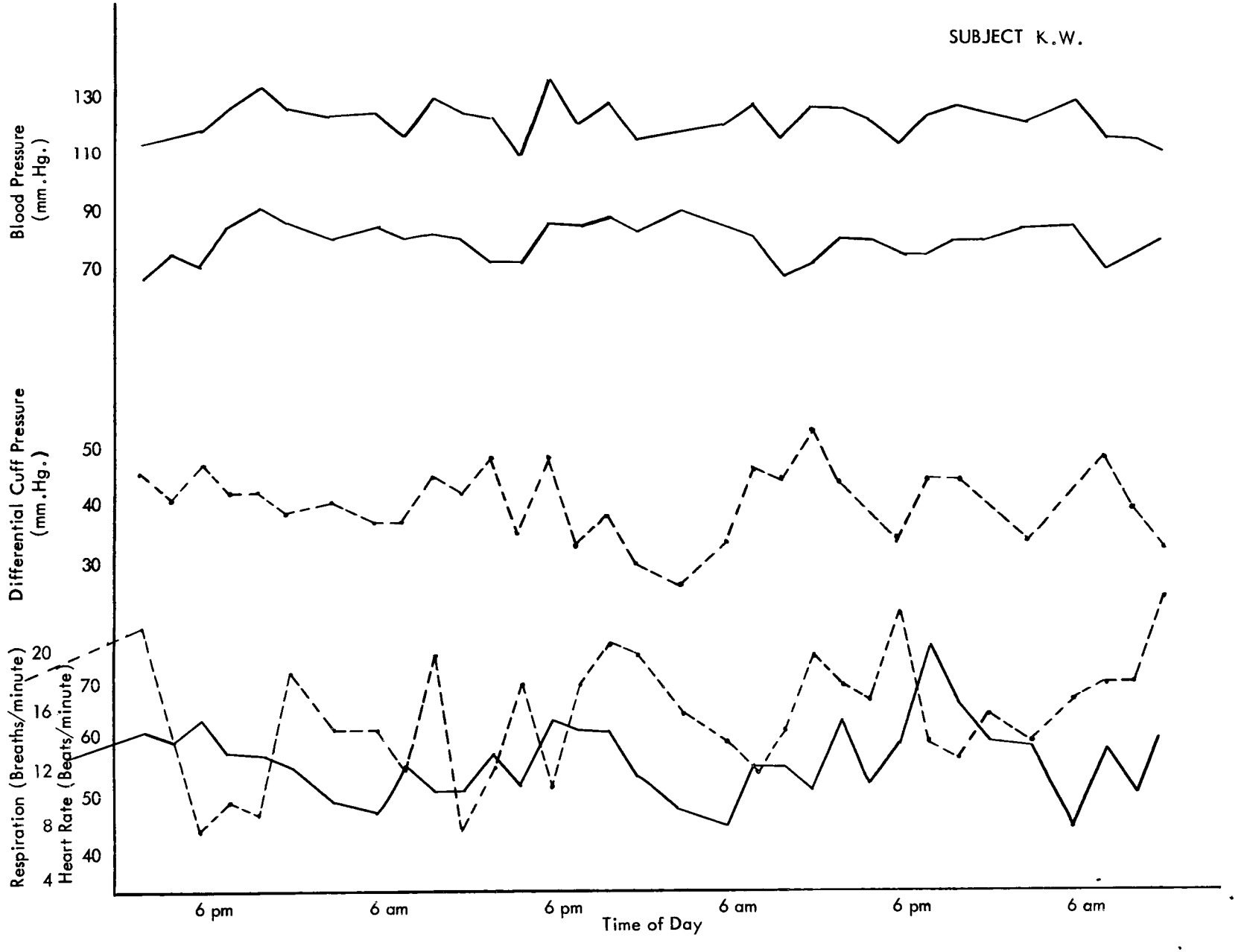


Subject J.P. Figure 11. Vital signs -- 72-hour bedrest study with the Flack Maneuver.

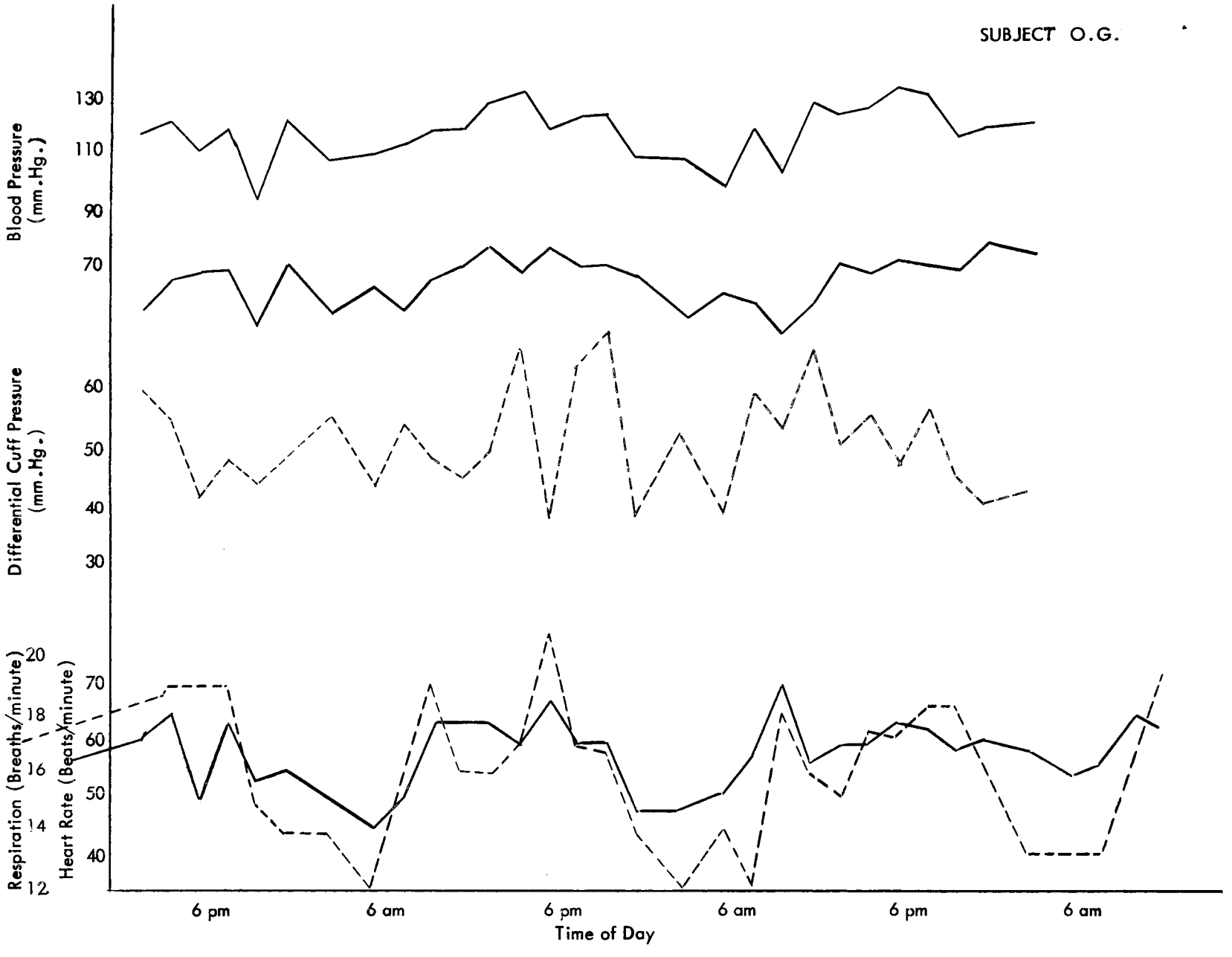


Subject K.W.

Figure 12. Vital signs -- 72-hour bedrest study.



Subject K.W. Figure 13. Vital signs -- 72-hour bedrest study with the Flack Maneuver.



Subject O.G.

Figure 14. Vital signs -- 72-hour bedrest study.

was attained. Response to the tilt procedure performed at the end of the first immobilization period showed greater heart rate increases compared to the pre-immobilization studies. Figures 15 through 20 show these heart rate changes. The relatively slow cycling of the cuff-microphone technique prevented acquisition of blood pressure measurements which were coincident to the time of occurrence of syncope, or which were representative of the dynamic changes in blood pressure during a Flack maneuver. The blood pressure measurements obtained were not plotted because of the little additional information they presented.

Some figures appear incomplete because data were not obtained on all subjects for all four tilt procedures, usually because of syncope or convulsions.

At the bottom of each figure is a graphic representation of the position of the tilt table at the time the heart rates were determined. The lines of the figures connect heart rate measurements which were determined as follows. The heart rate prior to tilt was determined by counting QRS complexes in a 15-second interval in the steady state condition prior to tilting, and then converted to beats per minute; the heart rate in the upright position was determined by counting the QRS complexes in the 15-second interval after the 70° tilt position was obtained, and then converted to beats per minute; the heart rates reported in response to the Flack maneuver were determined by counting 10-second intervals at the time that the maximum and minimum heart rates occurred and converted to beats per minute. Each group of four lines represents heart rate changes for comparable times in the tilt procedure in the pre-bedrest and post-bedrest evaluations made in association with the two study periods. Thus the first group of four lines is found directly above the graphic illustration which indicates a change in the position of the tilt table from 0° to 70°. The first point in each line in the second group of lines represent the heart rate immediately prior to tilt down. The end point in the line represents the heart rate in the horizontal position. Heart rate changes that are associated with performance of the first Flack maneuver are indicated next. The first point represents the heart rate immediately prior to tilt; the second point, the maximum heart rate during the Flack test; and the third point, the value obtained in the period of rebound slowing of the heart. The second and third values reflect increased heart rate from tilting as well as the Flack maneuver. The first point in this group, which was obtained prior to tilt, is presented to provide an indication of the comparative heart rates immediately prior to each tilt.

A review of figures 15 through 20 shows that the heart rate changes in response to tilting from 0° to 70° were greater after bedrest than before bedrest. Similar degrees of change of heart rate with tilting were noted after the second bedrest period (to which periodic Flack maneuvers were added) compared to the period of bedrest alone. The heart rate changes noted in response to tilts performed before the second study period were greater than those obtained in tilts before the first study period.

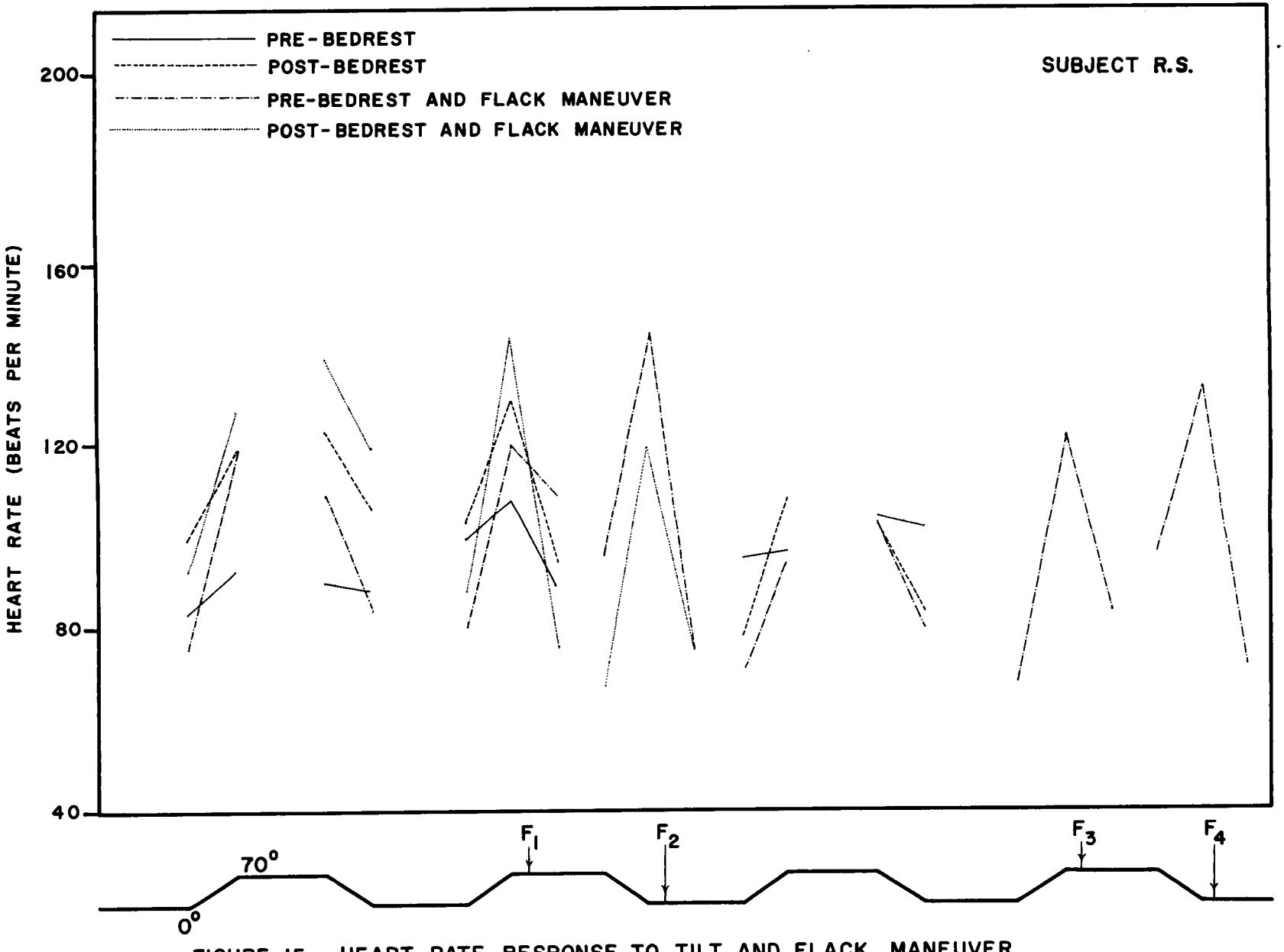


FIGURE 15. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

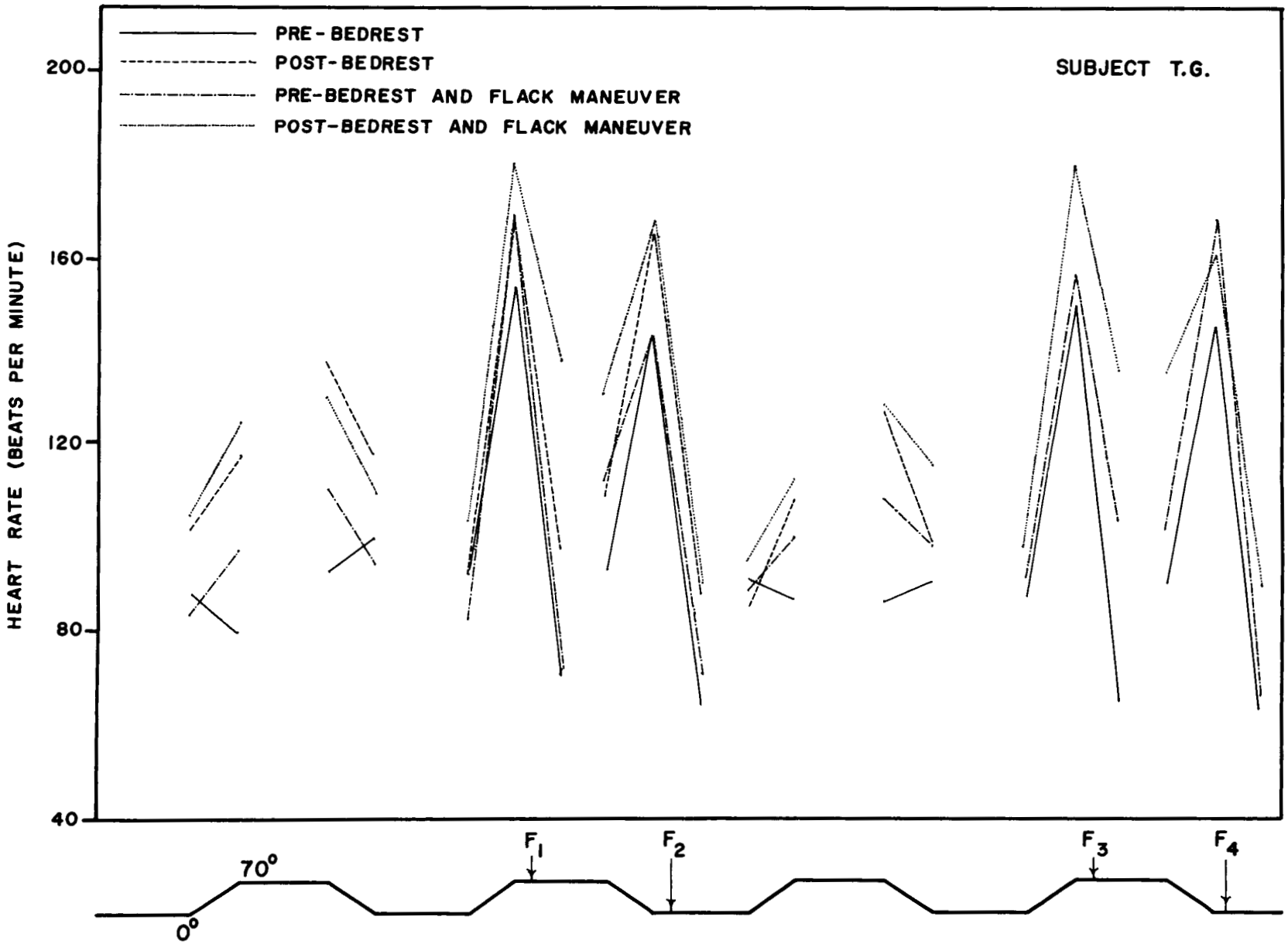


FIGURE 16. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

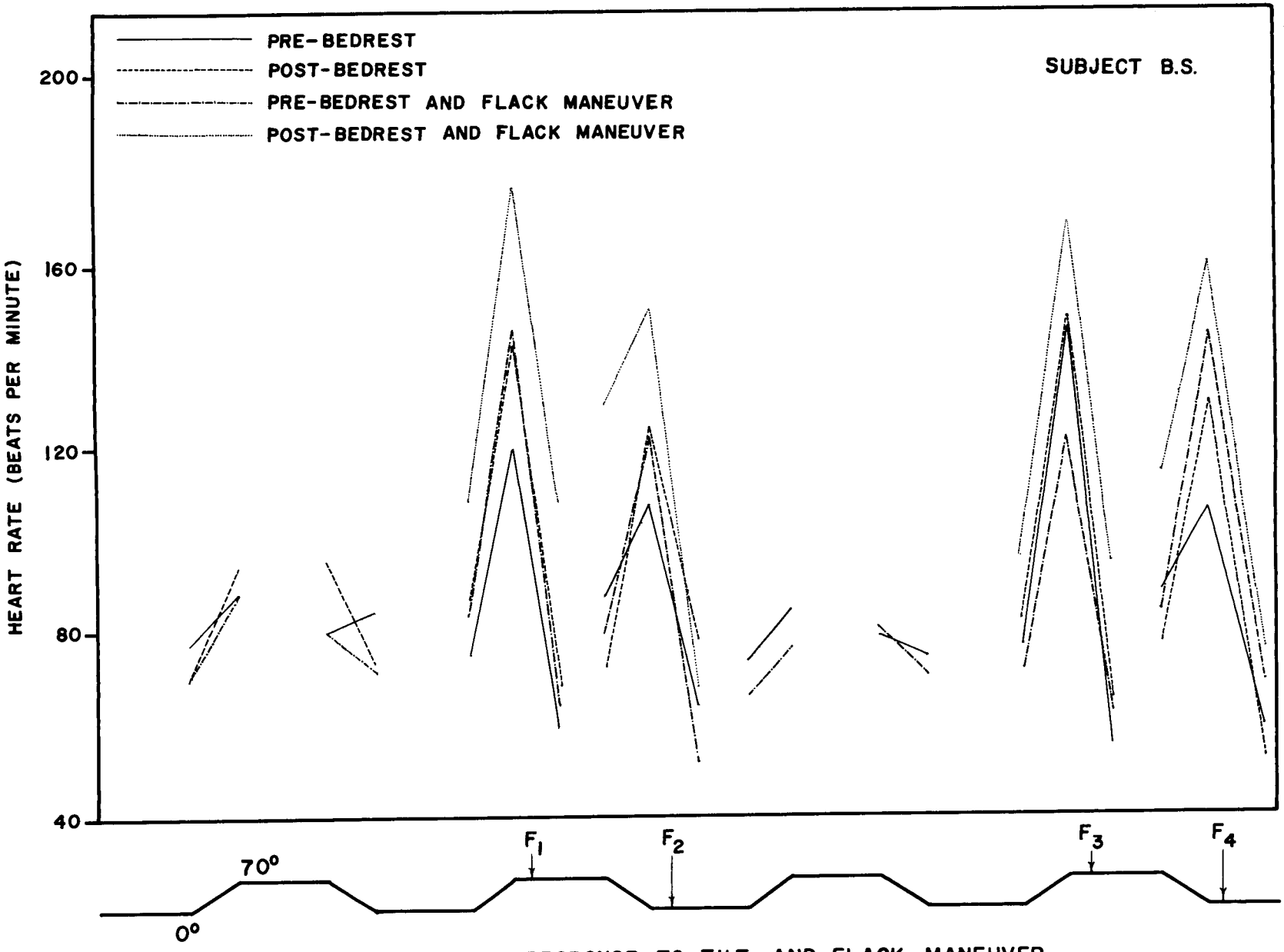


FIGURE 17. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

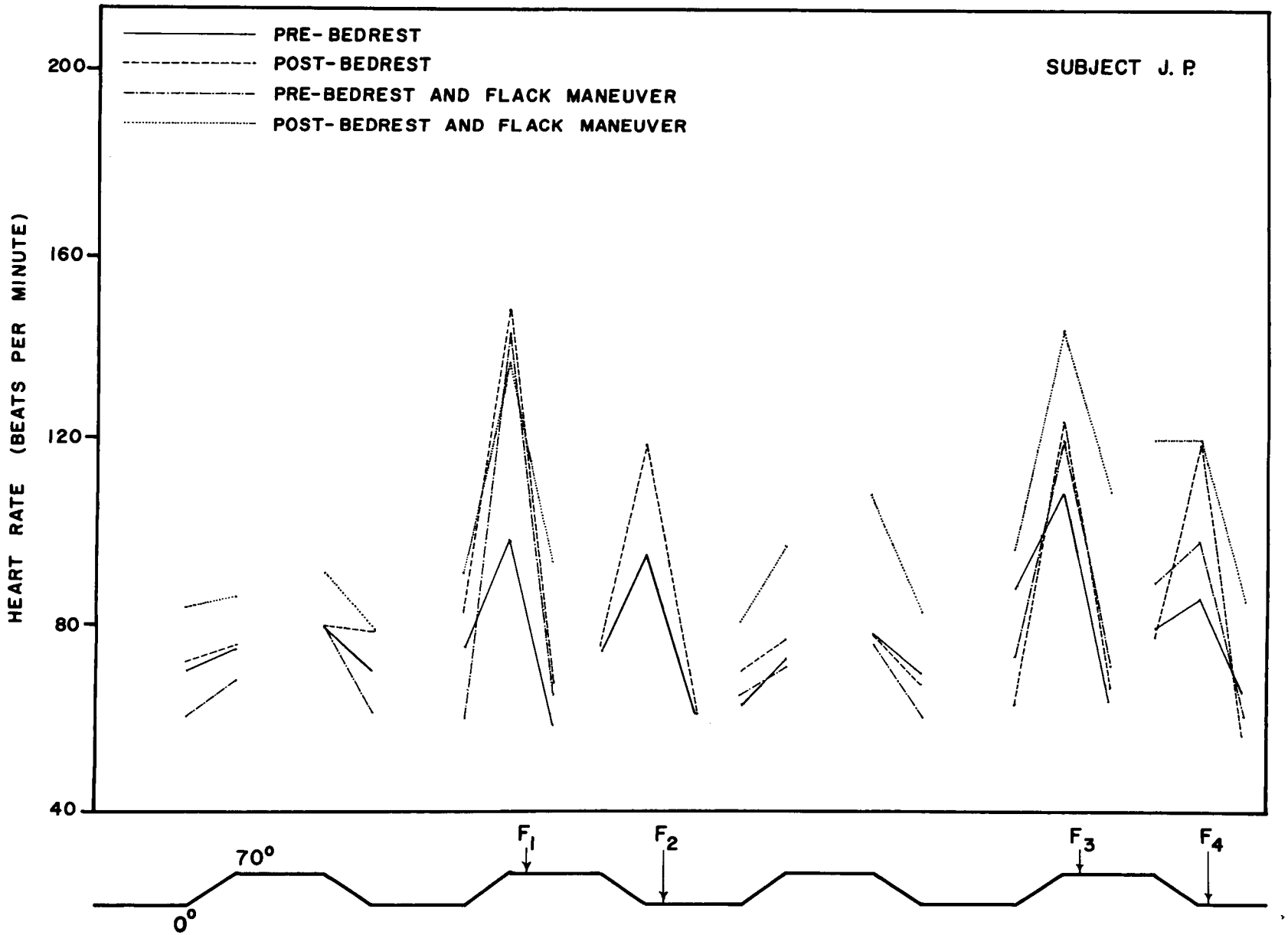


FIGURE 18. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

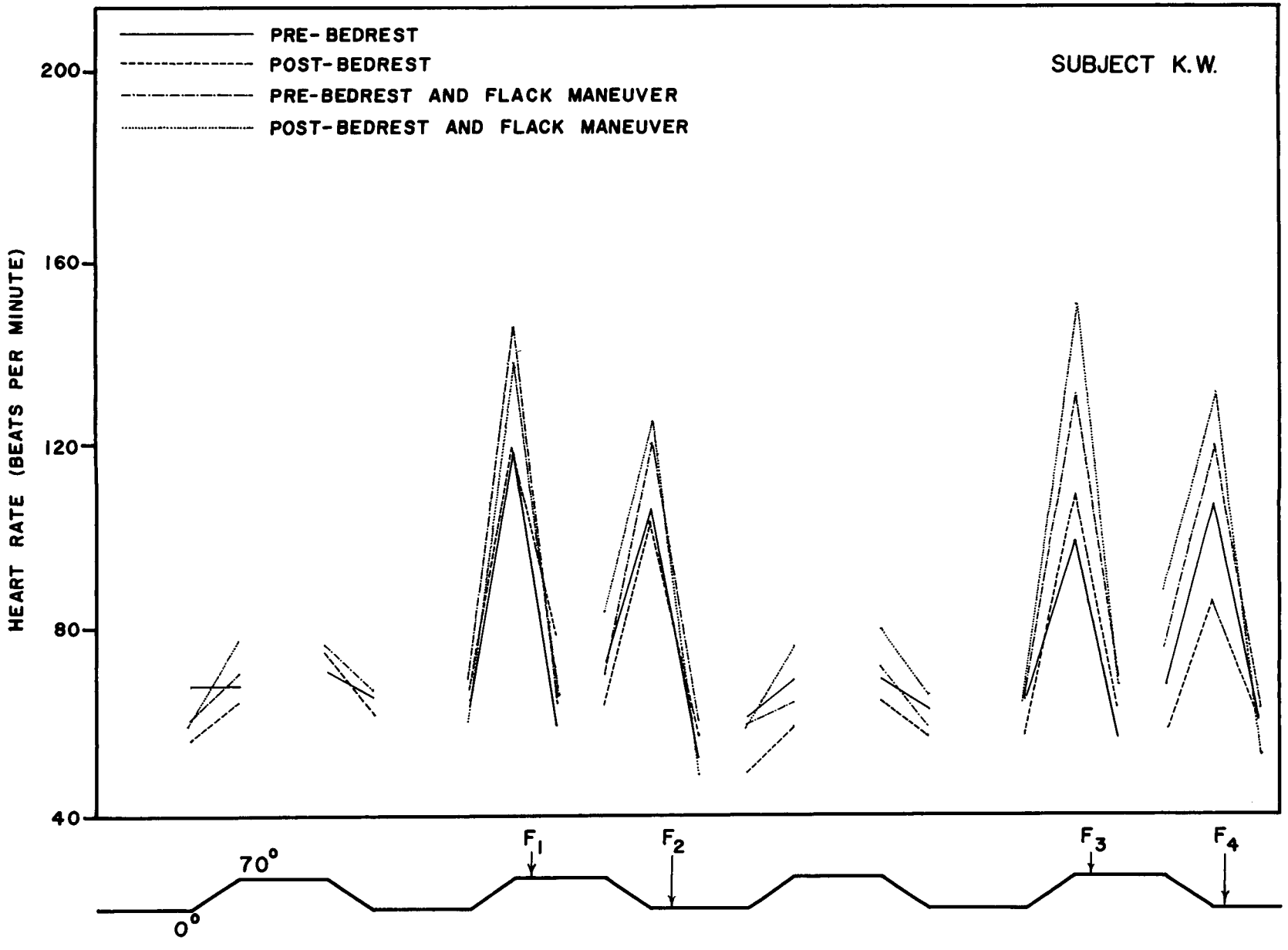


FIGURE 19. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

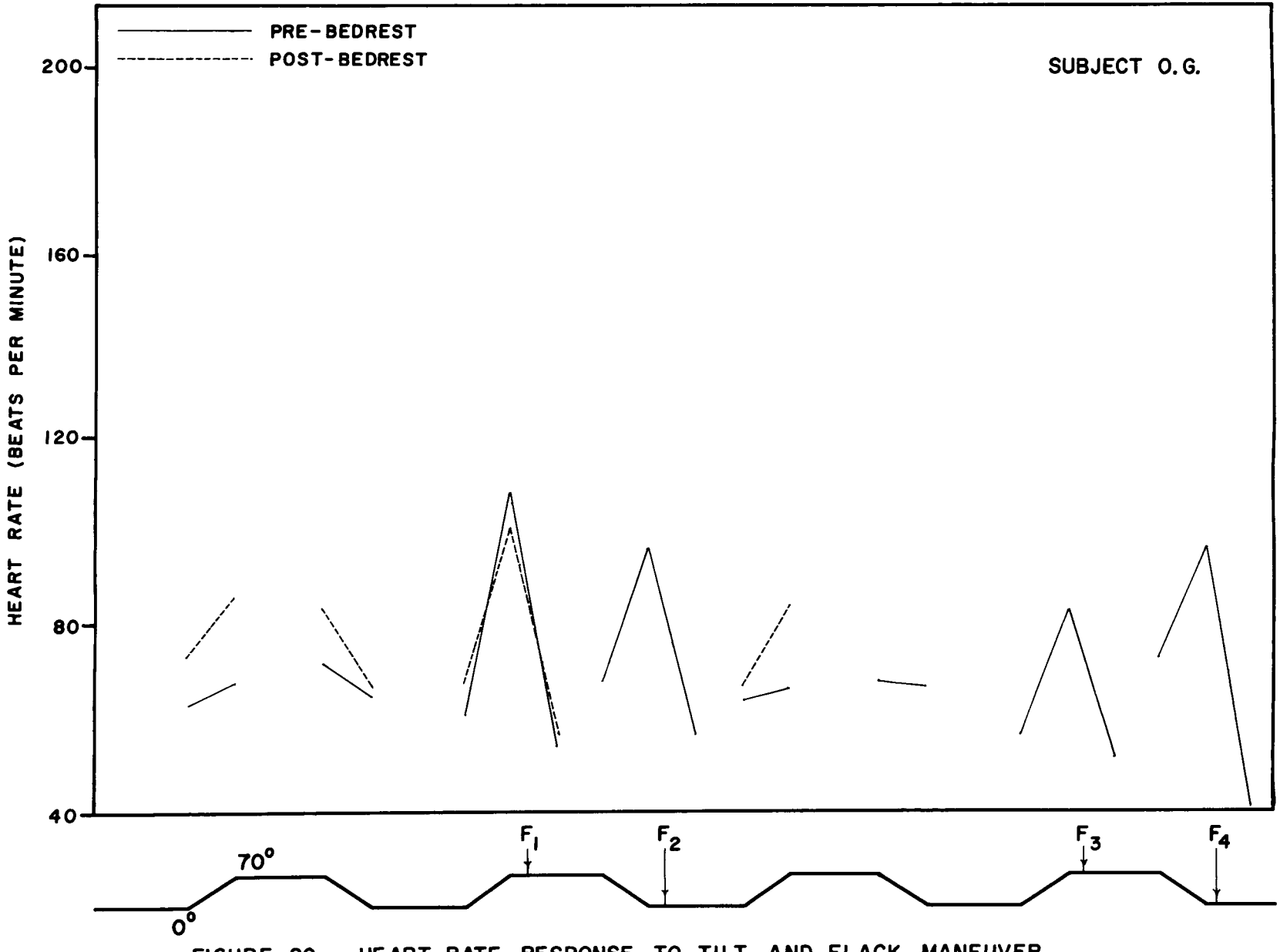


FIGURE 20. HEART RATE RESPONSE TO TILT AND FLACK MANEUVER

The maximum heart rates in response to the Flack maneuver in the 70° tilt position were greater after bedrest than before bedrest. A similar finding was observed for the study to which periodic Flack maneuvers were performed during the bedrest period. The maximum heart rates in response to the Flack maneuver were greater prior to the second period than prior to the first period.

Subjects T.G., R.S., and O.G. experienced convulsive syncope during performance of the first Flack maneuver in the 70° tilt position after the first period of bedrest. Blood pressure measurements were not available at the exact time of the convulsive episodes because of the infrequent cycling (every 30 seconds) of the cuff-microphone blood pressure apparatus. But, the measurements preceding the episode showed systolic blood pressures of approximately 90 mm Hg and diastolic pressure of approximately 75 mm Hg. Figure 21 shows the record made on subject O.G. at the time of his convulsive syncope episodes. His convulsion occurred shortly after the start of the interval in which there was no cardiac activity for an 8-second period. There was a slowing of the heart rate prior to the period of asystole. Subject B.S. experienced dizziness while performing the first Flack maneuver following bedrest. Subject T.G. experienced dizziness during the first Flack maneuver of the tilt prior to the second bedrest period. Subjects R.S. and T.G. experienced dizziness during the first Flack maneuver after the second period of bedrest.

Table I presents the heart rate changes observed during passive tilt procedures before and after the two study periods of recumbency. The steady state heart rate prior to tilting to the 70° position is shown in parentheses. The heart rate change represents the difference between the steady state rate observed in the supine position, and the maximum rate obtained in the 70° position.

All six subjects showed heart rate increases of less than 10 beats per minute to the initial tilt studies. Following bedrest all subjects showed a larger increase in heart rate with tilting, with increases ranging from 10 to 44 beats per minute. The steady state heart rate in the supine position tended to be higher after bedrest than before bedrest, but this was not true in all subjects for both bedrest periods. The heart rate changes to the tilt prior to the second bedrest period were greater than prior to the first bedrest period, and in some cases were greater than those obtained immediately after the first period of bedrest.

DISCUSSION

The term "cardiovascular deconditioning" has been used to describe the postural changes in heart rate, blood pressure, and perhaps other not yet well identified changes in cardiovascular dynamics that are observed after bedrest, water immersion, and weightlessness. At present this cardiovascular deconditioning is thought to result from a combination of mechanisms.

Because of the insufficient knowledge about these mechanisms, the interpretation of measurements of heart rate and blood pressure becomes difficult. We

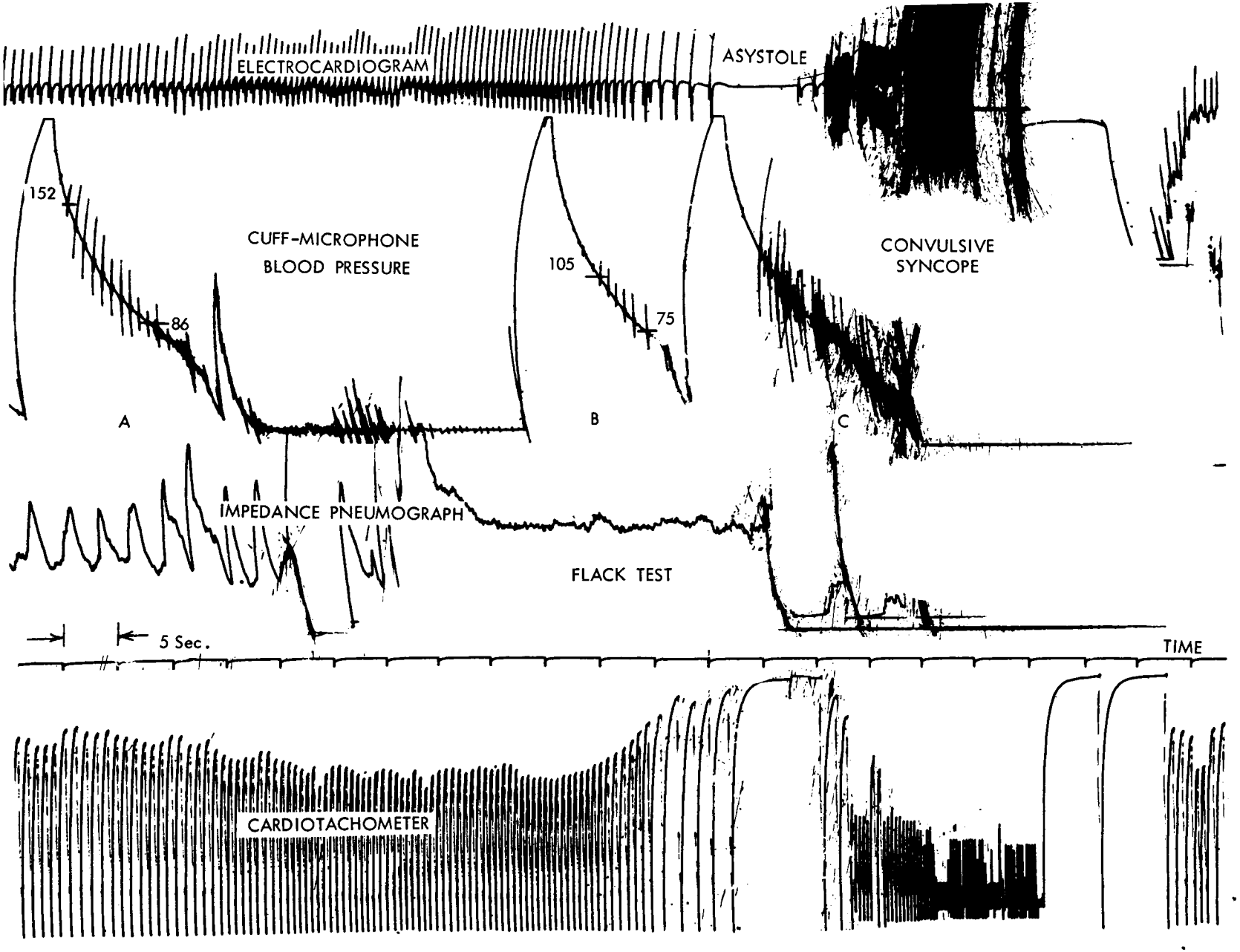


Figure 21. Cardiac asystole and convulsive syncope.

TABLE I

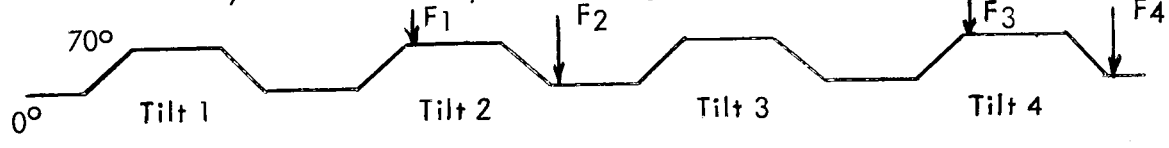
HEART RATE CHANGES TO PASSIVE TILT PROCEDURES

(Beats/Minute)

Subject	Period	Pre-Bedrest				Post-Bedrest			
		Tilt 1		Tilt 3		Tilt 1		Tilt 3	
R.S.	1	8	(84)	0	(96)	20	(100)	28	(80)
	2	44	(76)	30	(74)	44	(96)	--	--
T.G.	1	6	(88)	0	(94)	34	(104)	22	(86)
	2	28	(84)	28	(80)	24	(106)	32	(96)
B.S.	1	10	(78)	10	(74)	22	(74)	--	--
	2	16	(72)	14	(66)	32	(100)	22	(88)
J.P.	1	8	(74)	10	(64)	10	(74)	10	(70)
	2	19	(61)	12	(64)	8	(84)	26	(82)
K.W.	1	4	(68)	6	(64)	20	(56)	10	(56)
	2	14	(62)	12	(60)	18	(64)	18	(60)
O.G.	1	4	(64)	2	(64)	18	(68)	--	--
	2	--	--	--	--	--	--	--	--

Period 1 = Bedrest Period 2 = Bedrest plus periodic Flack maneuvers

Steady state heart rate prior to tilting is shown in parentheses.



F_1, F_2, F_3, F_4 = Time of performing Flack maneuver

cannot quantitate confidently the severity of deconditioning from resting heart rate, maximum heart rate attained during tilt, or the changes in heart rate during tilt. At present, we are limited to qualitative descriptions of the severity of deconditioning by considering the interrelation of the several measurements made. Consideration is given to heart rate, heart rate changes, blood pressure, pulse pressure, and appearance of the subject. It is not known whether the degree of deconditioning is reflected more accurately by the rapidity with which syncope occurs or by the pattern of changes in blood pressure and heart rate prior to its occurrence.

The Flack maneuver as used in these studies is modified from the maneuver described by Flack,[18] and can be considered as a controlled Valsalve experiment. Both the Flack and Valsalva maneuvers have been used to evaluate the circulatory integrity or efficiency.[19,20,21,22,23,24,25,26,27,28,29,30] A modified Valsalva maneuver, the M-1 maneuver, has been used[31,32] to overcome undesirable hemodynamic effects during exposure to high positive acceleration. Transfer of fluid from the blood (vascular space) to the extravascular space has been shown by Brown[33] to occur from the performance of repeated Valsalva maneuvers. As far as could be determined, no studies have been conducted to evaluate the conditioning effect of periodic performance of such respiratory maneuvers during prolonged periods of bedrest.

The occurrence of convulsive syncope in three subjects during performance of the Flack maneuver in the 70° tilt position after three days of bedrest can be interpreted as evidence of cardiovascular deconditioning resulting from relatively short periods of recumbency since it did not occur before bedrest. In the tilt procedure of the second bedrest study, the subject was tilted down at signs or complaints of impending syncope. For this reason, the failure to observe convulsive syncope after the second period of bedrest is not an indication of a lesser degree of deconditioning than observed after the first study.

The maintenance of cardiovascular conditioning or adaptability is of importance to functioning of all body systems, for without blood pressure and flow there cannot be continued performance of these systems. Syncope is but the extreme manifestation, or end point, describing the lack of cardiovascular adaptability as far as the central nervous system is concerned; it indicates the extreme dependence of the central nervous system on oxygenation that comes with blood flow. Table II presents a classification of the mechanisms which may have been responsible, singularly or in combination, for the syncope observed in these subjects.

The mechanism of convulsive syncope in one subject was probably the systemic hypotension that resulted after cardiac arrest (see Figure 21) of 8 seconds duration. Such arrhythmias in relation to respiratory maneuvers have been described,[19,31,32,34,35,36] and in fact two papers report asystole for 3 to 12 seconds duration.[31,32] Cardiac asystole could have resulted from excessive vagal activity suggested by the slowing of the heart prior to asystole.

TABLE II

MECHANISMS OF SYNCOPE

1. Cardiac (Primary mechanism results from inadequate performance of the heart)
   a. Asystole
   b. Valvular
   c. Myocardial
   d. Cardiac compression
   e. Arrhythmia

2. Venous (Primary mechanism results from inadequate return of blood to the heart)
   a. Loss of venomotor tone
   b. Venous pooling; systemic or pulmonary
   c. Increased resistance to flow (respiratory pressure maneuvers)
   d. Lack of muscle tone

3. Arteriolar (Primary mechanism results from inability to maintain blood pressure)
   a. Loss of reflex mechanisms
   b. Changes in distribution of circulating blood

4. Effective circulating blood volume decreases (With resulting decrease in cardiac output)
   a. Loss of blood
   b. Pooling of blood
      1) Venous pooling
      2) Arteriolar change in distribution
   c. Loss of fluid to extravascular space

5. Neurological (Primary central nervous system mechanisms)
   a. Reflexes
   b. Localized disease
   c. Vasovagal (? trigger)

The convulsive syncope in the other two subjects probably resulted from systemic hypotension and brain hypoxia that occurred during the performance of the Flack maneuver. This type of syncope has been described in detail in other presentations.[37,38,39] A discussion of the physiological relationships between intrathoracic, intraspinal, and intra-arterial pressures is covered by Hamilton.[40] Cardiac compression,[41] during the Flack test, must be considered as a possible contributing factor. Evidence that would indicate myocardial deterioration has been implied in observation from space flight.[42]

Venous mechanisms assume a most important role in the return of blood to the heart.[43,44,45,46,47,48] Loss of venomotor tone is suggested from the observation of the dependent veins on Astronaut Schirra[11] but superficial venous engorgement was not prominent in these studies at the Texas Institute for Rehabilitation and Research. Venous pooling in the legs that normally occurs due to the increased hydrostatic column of the erect position likely played some role. The relation of venomotor tone[49] and venous pooling is obvious. Increased pulmonary vascular resistance to flow likely resulted during the performance of the Flack test.[23,24]

Involvement of arteriolar reflex mechanism, seen in classical orthostatic hypotension[50,51,52,53,54,55] has been suggested as possibly occurring if the gravitational force is reduced or maintained in a constant direction.

The effective circulating blood volume is closely related to the venous control mechanisms. Loss of effective circulating volume can occur by venous pooling described above. The transfer of fluid from the vascular system to the extravascular space of the legs occurs in normal persons[56,57,58,59,60,61,62,63] upon assuming the erect posture and is likely to be accentuated after prolonged bedrest or weightlessness because of a relative deficiency of interstitial fluid, which had transferred to the vascular system (and then diuresed) during loss of exposure to gravitational forces. In addition, changes in blood volume have been noted with prolonged recumbency.[1,2,4,64,65]

Central nervous system factors can be incriminated directly, as well as indirectly through changes in blood pressure. Of primary concern is the vasodepressor or vasovagal type of syncope[66] seen in normal subject (in response to a variety of stimuli).

The syncope seen in these experiments likely resulted from a combination of the factors listed, especially since it occurred during tilt in association with the Flack maneuver. But since the syncope only occurred after, and not before bedrest, there is an indication that cardiovascular deconditioning did occur, and was made manifest by this provocative procedure.

Greater heart rate changes to tilting and to the Flack maneuver after bedrest, compared to before bedrest, can also be interpreted as evidence of

cardiovascular deconditioning, since it was during these same tilt procedures that convulsive syncope was observed. Using this criteria, one can interpret the heart rate changes during the tilt procedure prior to the second period of bedrest as evidence of residual deconditioning from the first bedrest period Such residual deconditioning is conceivable because of the short time interval between the periods of recumbency. Consideration of the heart rate during the tilt procedures does not indicate any lessening of the degree of deconditioning. In fact, there are larger changes after the second period in several subjects compared to the first period. But, the heart rate findings after the second period of bedrest were more pronounced than the findings during the tilt prior to the second period of bedrest. Thus, interpretation of the effect of repeated Flack maneuvers during bedrest is difficult because of the possibility of some residual deconditioning from the first bedrest period. There is evidence of further cardiovascular deconditioning during the second bedrest period, but one cannot say whether this is more or less than would have occurred had the Flack maneuver not been performed. It can be said only that the Flack maneuver did not totally prevent the cardiovascular deconditioning of bedrest.

An analysis of the vital signs obtained during the periods of recumbency revealed both individual and group variability which points to the difficult of interpreting the significance of a single measurement on a subject. The circadian pattern of change was quite distinct in some subjects. Upon a visual insepction there was no apparent correlation of the measurements obtained during bedrest with the tilt response after bedrest.

CONCLUSIONS

1. Cardiovascular deconditioning, manifested by an accentuated heart rate increase and convulsive syncope during performance of the Flack maneuver in the upright position, occurs after 3 days of bedrest immobilization.

2. Physiological measurements made during the bedrest immobilization and during tilt table studies do not indicate that the Flack maneuver, as performed for 30 seconds, every 30 minutes, prevents cardiovascular deconditioning that occurs during prolonged bedrest.

3. Preliminary studies point to the potential usefulness of the Flack maneuver as a provocative test to detect early cardiovascular deconditioning.

4. Overt convulsive syncope and cardiac asystole in association with the Flack maneuver makes its use undesirable in flight, or as a provocative test after flight if there are marked orthostatic changes in heart rate.

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